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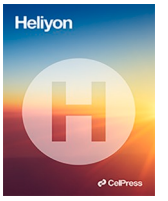

Case report

TAFRO syndrome with abdominal pain as the first symptom accompanied by liver damage with hyperbilirubinemia: A case report

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ABSTRACT

Thrombocytopenia, anasarca, fever, reticulin fibrosis on bone marrow biopsy/renal dysfunction, and organomegaly (TAFRO) syndrome are infrequent conditions with diverse clinical and pathological characteristics related to multi-organ damage. There are few reports of TAFRO syndrome accompanied by liver damage with hyperbilirubinemia. We describe the case of a 61-year-old male who presented with sudden onset abdominal pain accompanied by liver damage with hyperbilirubinemia. His symptoms worsened, leading to fever, hepatic insufficiency, serous cavity effusions, thrombocytopenia, and acute renal failure. Fever and anasarca relapsed after steroid discontinuation. The patient was ultimately diagnosed with TAFRO syndrome by biopsies taken from the axillary lymph nodes. He was then administered steroids, which resolved his symptoms almost completely. Our case was notable for its atypical signs and total remission of TAFRO syndrome.

1. Background

TAFRO syndrome is a clinical subtype of idiopathic multicentric Castleman disease (iMCD) that is characterized by thrombocytopenia (T), anasarca (A), fever (F), reticulin fibrosis/renal insufficiency (R), and organomegaly (O) [1]. TAFRO syndrome was first described in 2010 [2]. The diagnostic criteria for TAFRO syndrome were published in 2021 [3]. For TAFRO syndrome, high-dose glucocorticoid medication is the recommended course of treatment. If this treatment is ineffective, second-line treatments include cyclosporine, tocilizumab, and rituximab. The disease is extremely rare, and physicians are not well-versed in treating it. It is challenging to diagnose the disease promptly and treat patients as soon as possible. We encountered a 61-year-old man who experienced liver damage with hyperbilirubinemia in addition to the core signs. After four months of hormone treatment, he experienced notable improvement in his condition. However, he experienced fever and edema eight months after the cessation of hormone therapy. Subsequently, the TAFRO diagnosis was confirmed by lymph node biopsy. His clinical symptoms and radiological and laboratory findings significantly improved following one year of hormone treatment.

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2. Case presentation

A 61-year-old Chinese man was admitted to our hospital on July 3, 2021 because of poor appetite, lassitude, gastric distention and yellow urine for more than 2 weeks. He denied a history of medical or surgical illnesses. He had a 20-year history of alcohol consumption (average 20–40 g of alcohol per day) but no cigarette smoking. The patient experienced intermittent abdominal pain on March 12, 2021. A gastroscopy examination revealed chronic superficial gastritis with gastric erosion, *Helicobacter pylori* positivity (HP+), and multiple polyps of the colon. Tests that were negative for the 13C-urea breath were performed after two weeks of eradication medication. The patient underwent colon polypectomy on May 12, 2021, and his blood routine, liver function, and coagulation function were normal during hospitalization. He received the first dose of the COVID-19 vaccine on June 11, 2021. The patient subsequently developed yellow urine accompanied by chills on June 15, 2021. He experienced intermittent upper abdominal pain accompanied by radiating pain in the right back, nausea, fatigue, and poor appetite on June 18, 2021. The laboratory examination indicated serious abnormalities in liver function, with a rise in total bilirubin ten times the upper limit of normal. The patient was diagnosed with cholecystitis, biliary tract infection, and suspected drug-induced liver damage at an outside hospital. Following anti-infective and hepatoprotective treatment, the patient's infection symptoms worsened, he acquired increasing jaundice and rising bilirubin levels, coagulation dysfunction occurred, renal function declined, and multiple serosal cavity effusions and hypoalbuminemia manifested. The patient was referred to the Department of Liver Intensive Care Unit on July 3, 2021.

We performed a physical examination of the patient upon admission. The patient's vital signs were as follows: temperature, 36.5 °C; blood pressure, 128/87 mmHg; heart rate, 78 beats/min; and respiratory rate, 18 breaths/min. Physical examination revealed yellow skin, icteric sclera, reduced breath sounds in the bilateral lower lung areas, a distended abdomen with no discomfort or rebound pain, and suspected shifting dullness.

When he was admitted to our hospital, he was fever-free. Laboratory tests revealed severe hepatitis (total bilirubin, 361.2 μmol/L), hypoalbuminemia (24 g/L), renal failure (serum creatine, 140.86 μmol/L), elevated CRP levels (96.21 mg/L), high D-dimer levels (8280 μg/dl), high interleukin-6 (IL-6) levels (85.3 pg/μl), and a high ferritin level (1033 ng/ml). The patient tested HIV negative. The results of other laboratory tests are shown in Table 1. Fig. 1 displays the dynamic changes in the patient's chest CT scan. A modest pleural effusion was visible on both sides of the chest on the CT scan upon admission (Fig. 1A). After receiving hepatoprotective and antibacterial medication for one week, the patient's liver function improved. Nevertheless, the presentation is clinically different from that of classic liver disease. The patient's condition gradually deteriorated, with symptoms including lymphadenectasis, fever, thrombocytopenia, elevated total bilirubin, worsening renal function, persistent coagulation disturbance, subcutaneous edema, pericardial effusion, peripheral edema, pelvic, peritoneal, and pleural effusions. We scheduled several tests to arrive at the ultimate diagnosis. The results of next-generation sequencing (NGS) of peripheral blood and pleural fluid all revealed cytomegalovirus (CMV) (peripheral blood: N = 62 reads, pleural fluid: N = 22 reads). However, the CMV DNA test from the blood sample was negative. Multiple blood, ascites and pleural fluid cultures yielded negative results. Bone marrow smears revealed that bone marrow hyperplasia was significantly active, granulocyte hyperplasia was slightly shifted to the left, the red blood system was reduced, the proportion of monocytes was relatively high, and platelets were scattered and visible. Leukemia immunophenotyping revealed no obvious immune phenotype cells. Pathology of the bone marrow tissue revealed pathological changes in the patients with myeloproliferative anemia, and the bone marrow reticular fiber were negative. Positron emission tomography-CT (PET-CT) revealed multiple areas of inflammation in both lungs with atelectasis, bilateral pleural effusion (Fig. 2A), abdominal and pelvic fluid accumulation (Fig. 2B), anemia (Fig. 2C), splenomegaly, subcutaneous edema, decreased cerebral cortex glucose metabolism, multiple mildly enlarged lymph nodes in the left upper lobe, mediastinum, bilateral armpits (Fig. 2D), para abdominal aorta, bilateral pelvic walls (Fig. 2E), and groin, and increased glucose metabolism in the systemic bone marrow (Fig. 2F). Nevertheless, the patient refused to undergo lymph node biopsy. The clinical course and changes in clinical parameter levels following treatment with antibiotics, methylprednisolone, globulin, albumin, diuresis, 14 rounds of CRRT, and plasma dialysis filtration are shown in Fig. 3. The usage and dosage of steroids and globulin are all marked in Fig. 3.

Table 1
Laboratory data on admission.

Test	Value	Reference range
White blood cell	13.65	3.50–9.50 × 10^9/L
Platelets	224	125-350 × 10^9/L
C-reactive protein	96.21	0–10.00 mg/L
Alanine aminotransferase	10	7.00–40.00 U/L
Aspartate aminotransferase	18	13.00–35.00 U/L
Total bilirubin	361.2	3.40–20.50 μmol/L
Albumin	24	40.00–55.00 g/L
Blood urea nitrogen	10.36	3.10–8.80 mmol/L
Creatinine	140.86	41.00–81.00 μmol/L
Prothrombin time	15.4	11.00–14.50 s
D-dimer	8280	0–500 μg/dl
Fibrinogen	7.45	2.00–4.00 g/L
Interleukin-6	85.3	≤5.40 pg/mL
HIV	(−)	(−)
Ferritin	1033	32.00–501.00 ng/ml

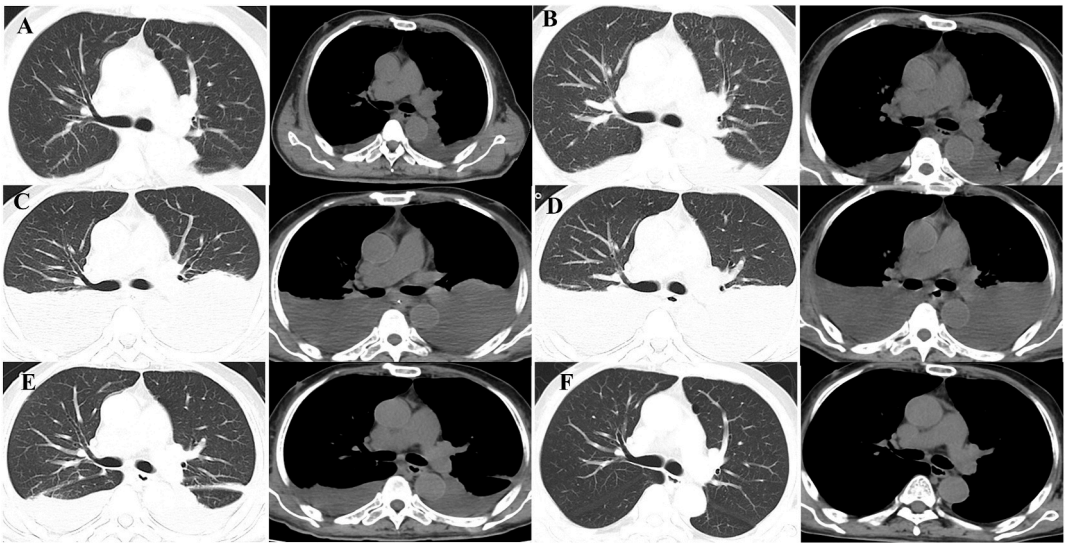

Fig. 1. Chest CT image. (A) Minor pleural effusion with partial atelectasis of both lower lungs on July 3, 2021. (B) Minor pleural effusion with partial atelectasis of both lower lungs on July 17, 2021. (C) Massive pleural effusion with partial atelectasis of both lower lungs on July 30, 2021. (D) Massive pleural effusion with partial atelectasis of both lower lungs on August 7, 2021. (E) Massive pleural effusion with partial atelectasis of both lower lungs on August 30, 2021. (F) The pleural effusion significantly decreased compared to that before on October 3, 2021.

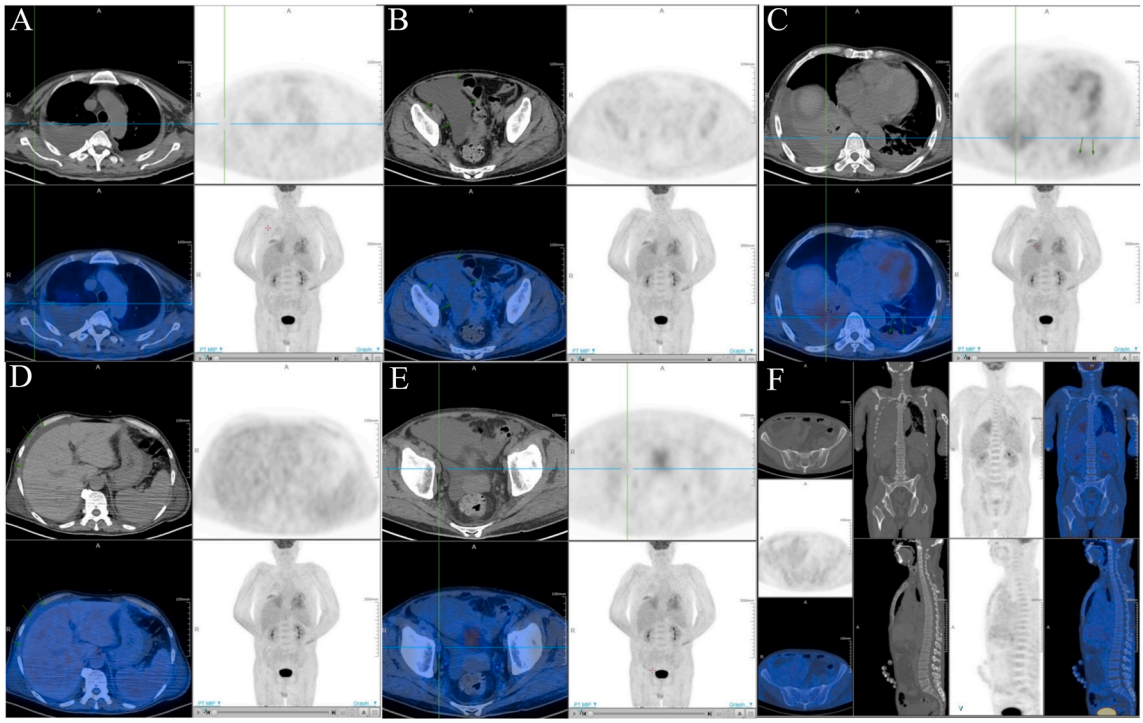

Fig. 2. PET/CT. (A) Pleural effusion. (B) Pelvic effusion. (C) Anemia. (D) Enlarged lymph node in the armpit. (E) Enlarged lymph node in the pelvic wall. (F) Increased glucose metabolism in systemic bone marrow.

Liver and kidney function gradually recovered, coagulation disorders improved, and hemoglobin and platelets progressively returned to normal levels. Multiple chest CT scans (Fig. 1B–F) revealed that the pleural effusion had greatly decreased from the previous CT scan results and was ultimately completely resolved. The patient was discharged on August 30, 2021 and was suspected to have TAFRO syndrome according to clinical guidelines [4]. The patient continued to receive oral hormone therapy after discharge. The patient underwent a diagnostic liver biopsy on September 7, 2021, and the liver tissues showed normal hepatocytes with no fibrosis or

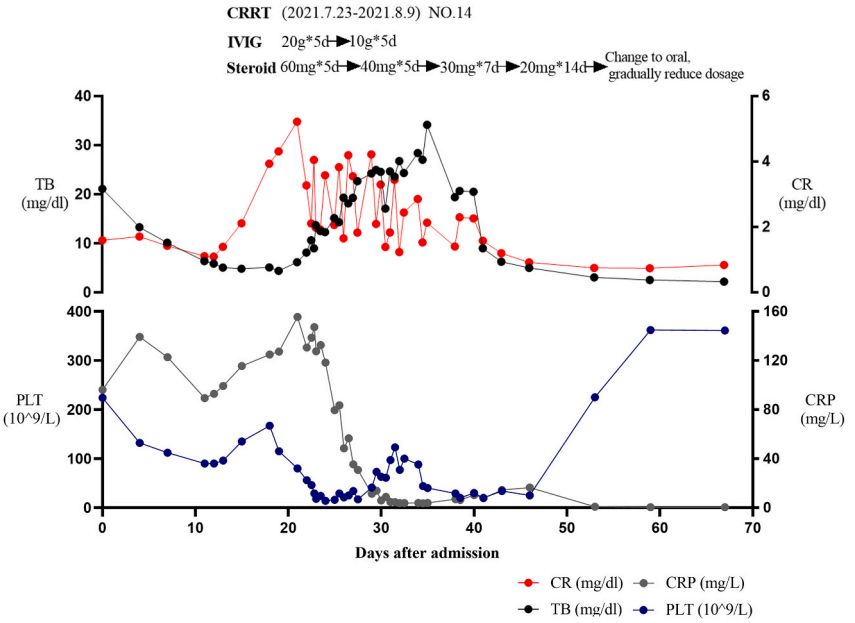

Fig. 3. The clinical course and changes in clinical parameter levels. CRRT Continuous renal replacement therapy; IVIG Intravenous immunoglobulins; TB Total bilirubin; PLT Platelets; CR Creatinine; CRP C-reactive protein.

noticeable morphological changes.

After four months of hormone therapy, the patient felt that his condition was stable; therefore, in October 2021, he stopped using oral hormones. On June 4, 2022, the patient experienced edema in both lower limbs, abdominal distension, and decreased platelets again. The patient was readmitted for treatment and underwent biopsy of a right axillary lymph node for pathological examination and immunohistochemistry. Afterward, other diseases, such as autoimmune diseases, connective tissue system diseases, infections, malignant tumors and tuberculosis, were excluded by all the clinical manifestations and auxiliary examinations, including immunohistochemistry (Fig. 4). The patient was diagnosed with Castleman's syndrome (Fig. 4A). All lymphocytes were positive for CD3 (T cells) (Fig. 4B), CD20 (B cells) (Fig. 4C), the proliferation marker Ki67 (Fig. 4D) and the proliferation marker CD138 (Fig. 4E) and were negative for HHV8 (Fig. 4F). Comprehensive assessment of clinical symptoms, laboratory results, and imaging findings revealed that the patient met the TAFRO syndrome diagnostic criteria. After undergoing hormone therapy once more, the patient's condition improved. Steroids were administered to the patient until June 2023. A recent phone follow-up revealed no recurrence.

3. Discussion

TAFRO syndrome is an infrequent disease, and it is a systemic inflammatory disease caused by a cytokine storm. The rapid course of the disease and the challenge of obtaining biopsy samples for diagnosis account for the high death rate associated with TAFRO syndrome. However, TAFRO syndrome-like symptoms can also be present in patients with a variety of other illnesses, making differential diagnosis challenging in patients with borderline disease. Our patient showed the typical clinical features of TAFRO syndrome, and the histological findings of his lymph nodes were compatible with those of iMCD. In addition to the usual symptoms, TAFRO syndrome can also present with a variety of manifestations; in this case, liver injury with hyperbilirubinemia worsened the condition.

A precise diagnosis is frequently challenging due to the phenotypic similarities between decompensated liver cirrhosis and TAFRO syndrome [5]. In the present case, the patient had a history of alcohol consumption. However, the patient's drinking history was not yet able to be diagnosed with alcoholic liver disease, and he did not have imaging or pathological evidence of cirrhosis. There are two reports of patients with hyperbilirubinemia [5,6], but a liver biopsy was not performed. There was one case of TAFRO syndrome with cholangitis on liver biopsy. However, review publications have not addressed hyperbilirubinemia or liver injury [1,7,8]. In our present case, CT revealed cholecystitis and no dilatation or filling defects in the intrahepatic bile duct or common bile duct. The patient had a history of alcohol consumption and had taken drugs suspected of causing liver damage. Although laboratory tests are not diagnostic for a specific etiology, we chose not to perform a liver biopsy during the period of high jaundice because of the risk of bleeding. Instead, we chose to perform liver biopsy during the recovery period. However, the liver biopsy results were basically normal, ruling out the pathological characteristics of chronic liver diseases, including autoimmune liver disease. Due to the increase in IL-6, vascular endothelial growth factor (VEGF), and other cytokines, which increase vascular permeability and cause resulting organ damage, TAFRO syndrome can cause systemic inflammatory responses. Despite the fact that our patient's serum IL-6 levels were more than ten times greater than the upper limit of normal, VEGF was not tested. In some critically ill individuals, the levels of inflammatory factors such as IL-6 gradually increase, causing cytokine storms [1]. We hypothesize that the unfavorable microenvironment created by the

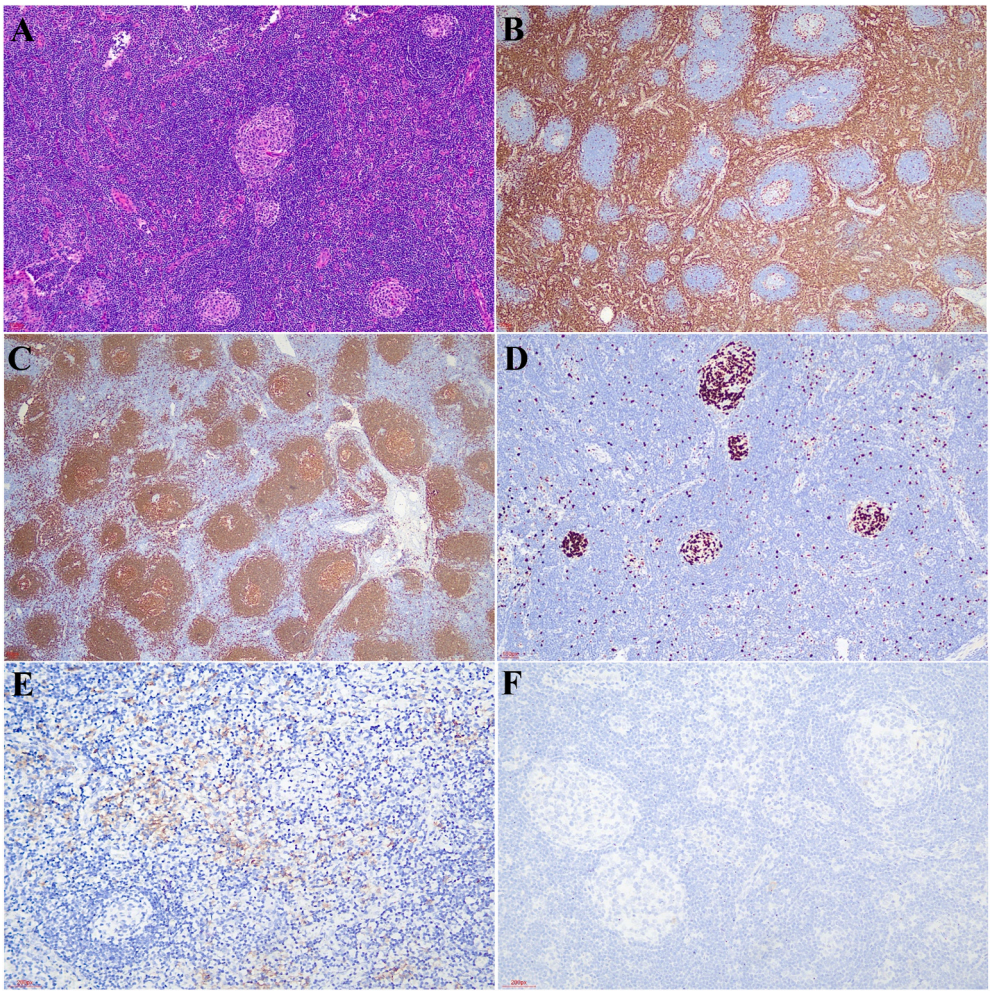

Fig. 4. Pathological and immunohistochemical results of the lymph node biopsy (right armpit) for Castleman disease. A: HE 10X; B: CD3 (+) 20X; C: CD20 (+) 20X; D: Ki67 (+) 10X; E: CD138 (+) 20X; F: HHV8 (−) 20X.

inflammatory cytokine storm is the primary factor contributing to liver damage in this patient with TAFRO syndrome. Liver tissue contains a considerable number of cells that participate in immunological responses and perform critical immune defense and regulatory activities [9]. Immune-mediated cytokine storms cause the production of a high number of pro-inflammatory molecules, which can increase non-specific immune inflammatory responses in the liver, resulting in secondary liver injury. The liver has a high demand for oxygen, making it vulnerable to the consequences of hypoxia. When there is high pleural effusion, respiratory failure, and renal failure in TAFRO syndrome patients, systemic arterial pressure and hepatic artery perfusion decrease, which can lead to hypoxic liver damage. When hypoxemia occurs, low oxygen and fat accumulation in liver cells can cause cell death, an oxidative stress response, the creation of several pro-inflammatory substances, and, eventually, liver injury. In vivo and in vitro studies have also proven that liver ischemia and hypoxia can promote liver cell death and inflammatory cell infiltration [10]. In our case, the patient was treated with the artificial liver technique, which led to rapid amelioration of hyperbilirubinemia. Moreover, hormones and internal medicine also play key roles in restoring liver function.

The patient's first symptom was abdominal pain. A recent systematic review revealed that abdominal pain in TAFRO syndrome patients is associated with adrenal abnormalities, such as adrenomegaly, adrenal ischemia/infarction or hemorrhage [11]. Previous research [12] has demonstrated that adrenal disorders on early-stage CT are effective for predicting unfavorable prognosis. Zhou Q et al. reported a case in which acute epigastric pain was the initial complaint, and computed tomography (CT) revealed retroperitoneal exudation surrounding the pancreas, resulting in a diagnosis of acute pancreatitis [13]. All ancillary investigations of this patient, including upper abdominal CT, positron emission tomography (PET)/CT, and abdominal ultrasound, revealed no adrenal or pancreatic abnormalities. In addition, his serum amylase and lipase levels were both normal. Abdominal ultrasonography revealed cholecystitis with gallstones. In conclusion, the gallbladder was identified as the source of the abdominal pain in this patient.

Although the etiology of TAFRO syndrome is unclear, its pathogenesis mainly involves excessive cytokine secretion and autoimmune dysfunction. Several experts speculate that MCD in immunocompetent patients might be due to proinflammatory

hypercytokinemia because of infection by a virus other than HHV8, inflammation, or neoplastic disease [12]. In our patient, although NGS revealed CMV infection, the number of sequences was small, and the serum CMV DNA result was negative; thus, we did not suspect that CMV was involved in the pathogenesis of this patient with TAFRO syndrome. Since the COVID-19 pandemic, there have been a few reports of TAFRO syndrome development following COVID-19 or COVID-19 immunization [14]. Cytokine storms play a role in both excessive immunoreaction after COVID-19 and TAFRO syndrome. Previously, three case reports [15–17] showed that TAFRO syndrome was triggered by the COVID-19 vaccine. One case of manifestations mimicking TAFRO syndrome can occur after COVID-19 [18]. Our patient was also vaccinated with the COVID-19 vaccine before disease onset. It is still unclear, however, whether the illness and the vaccination are connected.

TAFRO syndrome is a special subtype of iMCD. Its clinical manifestations, diagnosis and treatment are unique compared with those of other iMCD patients. The enlargement of lymph nodes for unknown reasons is suggested to improve the effectiveness of lymph node biopsy. In our patient, if he underwent a lymph node biopsy during the first hospitalization, he may not need to be hospitalized again.

An investigation [19] of 38 peer-reviewed case reports of TAFRO syndrome by Yamaguchi Y et al. revealed that the median recovery time from thrombocytopenia was 47.5 days. Our patient recovered from thrombocytopenia after 49 days, which aligns with earlier reported observations.

The standard of therapy for TAFRO syndrome has not been fully established; however, the most commonly applied treatments include IL-6 blocking therapy with siltuximab or tocilizumab and anti-inflammatory therapy with high-dose corticosteroids [20]. A recent study involving six patients with TAFRO syndrome indicated that early glucocorticoid therapy can improve prognosis, particularly in individuals with severe disease [21]. In 2019, Japanese researchers [4] proposed that individuals with clinical signs compatible with TAFRO but no pathologically proven Castleman disease be treated equally and vigorously. Therefore, we used hormone therapy in time. The patient's condition recurred after discontinuing steroids. The patient was diagnosed with TAFRO through lymph node biopsy and treated with steroids, to achieve a cure. The response rates to various treatment strategies for TAFRO syndrome vary, so personalized strategies should be highlighted [22].

According to a previous study, peritoneal dialysis is a viable treatment option for TAFRO syndrome patients who have acute renal injury and refractory ascites [23]. In our situation, peritoneal dialysis might also be attempted.

According to a recent Japanese study [24], D-dimer $\geq$18 μg/dL and age $\geq$60 years were found to be significant predictors of poor overall survival in TAFRO syndrome patients. Despite both of these risk factors, our patient's prognosis was favorable because of his vigorous therapy. To improve the patient's overall prognosis and quality of life, timely and precise diagnosis, efficient treatment, and recurrence prevention are critical.

4. Conclusions

TAFRO syndrome is rare and clinically complex. We encountered a patient with TAFRO syndrome who presented with manifestations similar to those of liver cirrhosis accompanied by liver damage with hyperbilirubinemia. The unusual symptoms and complete recovery of TAFRO syndrome in our study made it noteworthy. We would like to offer a reference for the diagnosis and treatment of this disease, promoting more research into this disease.

Ethics approval and consent to participate

The study was approved by the medical ethics committee of Shanghai Public Health Clinical Center. This study was conducted in accordance with the Declaration of Helsinki. Written informed consent for the publication of all images, clinical data and other data was obtained from the patient.

Data availability statement

No datasets were generated or analyzed during the current study.

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CRediT authorship contribution statement

Yu Liu: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Xue Mei:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Zhiping Qian:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Wei Yuan:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Jiefei Wang:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology,

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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